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THE ACTION OF CROSS CYLINDERS IN COMBINATION WITH OTHER LENSES.

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The Jackson Cross Cylinder which carries the name of the inventor is, I believe, one of the most valuable accessories of any trial case. Its intelligent application is extremely beneficial to all optometrists. It is a time saver and it insures accuracy. The most common forms of the cross cylinder are:

(1) —0.12 D. Sph ⊂ + 0.25 D. cyl.
(2) —0.25 D. Sph ⊂ + 0.50 D. cyl.
(3) —0.50 D. Sph ⊂ + 1.00 D. cyl.
(4) —0.75 D. Sph ⊂ + 1.50 D. cyl.
(5) —1.00 D. Sph ⊂ + 2.00 D. cyl.

The latter two are but very seldom used, and those most commonly used are the second and third. They are usually so constructed that the minus axis is distinguished with white dots, and the plus axis with red dots. This is obviouly advantageous for it affords at an instant glance the knowledge of whether the plus or minus is before the eye. They are set into rings with handles that are about two inches in length, at 45 degrees, so as to permit by a single twirling motion of the thumb and forefinger, the shifting of the lens from minus to plus, or from plus to minus. In working with cross cylinders ability to quickly transpose lens combinations is essential. All optometrists are familiar with lens transpositions, but cross cylinder transposition is very often confusing. It is for this reason that some of the following rules may prove of interest. Strange as it may seem, in all of our literature on this subject, except those dealing with dynamic cross cylinder tests, none give cognizance to the fact that the spherical as well as the cylindrical power is involved when the cross cylinder is used in the distance test. I have questioned a number of refractionists on this subject, and have been informed that only the cylindrical element is involved when the cross cylinder is employed. I think the subsequent facts will prove otherwise. Let it be understood that this is not a paper on cross cylinder procedure, but rather as the title states, "their action in

combination with other lenses." The following are the various types of errors that are encountered in refracting:

(1) Hyperopic errors corrected with plus spheres.

(2) Myopic errors corrected with minus spheres.

(3) Simple hyperopic astigmatism, corrected with plus cylinders.

(4) Simple myopic astigmatism, corrected with minus cylinders.

(5) Compound hyperopic astigmatism, corrected by the combination of plus spheres and cylinders.

(6) Compound myopic astigmatism, corrected with minus spheres combined with minus cylinders.

(7) Mixed astigmatism, corrected with minus spheres combined with plus cylinders, or the reverse. Some refractionists using plus, and others minus cylinders. I have found when using cross cylinders a good deal of confusion can be avoided, if only the plus element be considered. Therefore, throughout this paper, whenever reference is made to the axis of a cross cylinder, it will be understood that a plus cylinder axis 90, or a minus cylinder axis 180 is referred to.

Hyperopic Errors Corrected with Plus Spheres

We will assume that the lens in the trial frame on the patient's face is a plus 2.00 D. Sph. (In all of the following cases the —0.25 D. Sph ⊃ + 0.50 D. Cyl., cross cylinder is used.) The cross cylinder is placed in front of the trial frame with the axis at 90°. If vision is improved with the cross cylinder held in this position, it indicates, I believe, that there is a half diopter of uncorrected astigmia present in the vertical meridian, and also that the sphere must be reduced by a ¼ diopter, which makes the resultant prescription + 1.75 D. Sph ⊃ + 0.50 D. Cyl. axis 90. With the above combination replacing the former sphere in the trial frame, once more place the cylinder with its axis corresponding to the axis of the cylinder in trial frame. If on this trial vision is not improved, or if it becomes materially worse, shift the cross cylinder so that its axis is at right angles to the axis of the cylinder in the trial cell. If the vision is not improved, or becomes materially worse, remove cross cylinder entirely, if patient now reports vision is best without "other lens," you are assured your correction is + 1.75 D. Sph ⊃ + 0.50 D. Cyl. axis 90.

Let us assume that when the cross cylinder was placed over the sphere the patient's vision was not improved; the cross cylinder is then twirled so that the axis of the cross cylinder is at 180. If in this position vision is improved, it shows the necessity of reducing the sphere a ¼ diopter and placing a plus 0.50 D. cylinder axis horizontal in the trial cell. The formula is then + 1.75 D. Sph ⊃ + 0.50 D. Cyl. axis 180. From this point same procedure as before can be carried out.

Myopic Errors Corrected with Minus Spheres

We will assume that the lens in the trial frame is —2.00 D. Sph.

If, when placing the cross cylinder axis 90 in the trial frame the patient's vision is improved, a —0.50 D. cyl. axis 180, and reduction in the spherical element or ¼ diopter is indicated, the Rx being —1.75 D. Sph ⊃ —0.50 D. Cyl. axis 180. From this point, same procedure as in the first case, can be followed. If with the cross cylinder in the vertical position vision was not improved, but was found to be better when placed in the horizontal meridian, a —0.50 D. cylinder axis 90, and a reduction in the sphere of a ¼ diopter would be indicated. Let us now assume that in these cases, no matter how the cross cylinder was placed no improvement in vision could be noted, this would indicate that the spherical power alone was correct. To summarize the action of cross cylinders when used in conjunction with plus and minus spheres, let me say:

(a) When used in conjunction with any plus sphere, the spherical power is reduced a ¼ diopter, and a cylindrical lens of a ½ diopter is created, axis corresponding to the axis of the cylinder in the cross cylinder.

(b) When used in conjunction with any minus sphere, the spherical power is reduced a ¼ diopter, and cylindrical lens of minus ½ diopter is created, axis opposite or at right angles to axis of the cylinder in the cross cylinder.

Simple Hyperopic Astigmia Corrected with Plus Cylinders

Assuming that lens in trial frame is a + 1.00 D. Cyl. axis 90. When holding the cross cylinder axis corresponding to the axis of the lens in trial frame, and an improvement in vision is noted, a condition is evinced of —0.25 D. Sph ⊃ + 1.50 D. Cyl. axis 90. From this point same procedure can be carried on as in the previous cases. If, however, vision is not improved when cross cylinder is held axis 90, shift it so that axis is at right angles to lens in trial cell; if in this position vision is improved, a condition is present of requiring + 0.25 D. Sph ⊃ + 1.50 D. Cyl. axis 90. From this point the usual procedure can be carried on. To summarize the action on plus cylinder when used in combination with cross cylinders, let me say:

(a) When cross cylinder is held axis corresponding with the cylinder in the trial frame, the cylinder is increased a ½ diopter, and a minus 0.25 sphere is created.

(b) When cross cylinder is held, axis at right angles to the cylinder in the trial frame, the cylinder is decreased a ½ diopter, and a plus 0.25 sphere is created.

Simple Myopic Astigmatism Corrected with Minus Cylinders

Assuming that lens in trial frame is a — 1.00 D. Cyl. axis 180. When holding the cross cylinder axis corresponding with lens in trial frame, and improvement is noted in vision, the condition manifested is that the cylinder need be reduced ½ diopter, and that a — 0.25 D. sphere must be added, thus making the correction — 0.25

D. Sph ⊃ — 0.50 D. Cyl. axis 180. From this point the usual procedure can be carried on. Supposing when the cross cylinder was held in the above position, vision was either materially worse or was not improved. The cross cylinder would be twirled, the axis now at right angles to its previous position. If, in this position improvement in visual acuity was noted, it would show the necessity of increasing the cylinder ½ diopter, and adding a plus ¼ diopter sphere, thus making the correction as follows: + 0.25 D. Sph ⊃ — 1.50 D. Cyl. axis 180. To summarize the action of minus cylinders when used in conjunction with cross cylinders, let me say:

(a) When a cross cylinder is held axis corresponding with the minus cylinder in the trial frame, the cylinder is decreased a ½ diopter, and a minus ¼ diopter sph. is added.

(b) When cross cylinder is held axis at right angles to minus cylinder, the cylinder is increased a half diopter, and the sphere becomes of plus variety.

Compound Hyperopic Astigmatism Corrected by the Combination of Plus Sphericals and Cylindricals.

We will assume that the combination of lenses in the trial frame is a + 2.00 D. Sph ⊃ + 1.00 D. Cyl. axis 90. The cross cylinder is held with axis corresponding to cylinder in the trial frame. If vision is improved the necessity of reducing the sphere ¼ diopter and increasing the cylinder ½ diopter is manifested. The prescription then becomes + 1.75 D. Sph ⊃ + 1.50 D. Cyl. axis 90. Now let us suppose that vision was improved when the cross cylinder was twirled so that it was at right angles to the cylinders in the trial frame. This would necessitate increasing the sphere a ¼ diopter and decreasing the cylinder a ½ diopter. The formula would then be + 2.25 D. Sph. ⊃ + 0.50 D. Cyl. axis 90. To summarize the action of plus sphere and plus cylinder combinations when used with the cross cylinder, let me say:

(a) When the cross cylinder is held axis corresponding to the cylinder of the combination, the sphere is decreased and the cylinder is increased.

(b) When the cross cylinder is held axis at right angles to the cylinder of the combination, the sphere is increased and the cylinder is decreased.

Compound Myopic Astigmatism Corrected with Minus Spheres Combined with Minus Cylinders.

We will assume the lens combination in the trial frame before the patient's eyes is — 2.00 D. Sph. ⊃ — 1.00 D. Cyl. axis 180.

Placing cross cylinder axis corresponding to cylinder of the combination and if an improvement of vision is noted, the necessity for increasing the sphere ¼ diopter and decreasing the cylinder ½ diopter is manifested. If on the other hand, the improvement is noted when the cross cylinder is held at right angles to the cylinder

of the combination, it shows the necessity of decreasing the sphere and increasing the cylinder ½ diopter. Summarizing the action of minus spheres and minus cylinders used in combination with the cross cylinders, let me say:

(a) When the cross cylinder is held with its axis corresponding to cylinder of combination, the spherical power is increased ¼ diopter and the cylindrical power is decreased ½ diopter.

(b) When the cross cylinder is held with its axis at right angles to that of the cylinder in the combination, the spherical power is decreased and the cylindrical power is increased.

Mixed Astigmatism Corrected with Minus Spheres Combined with Plus Cylinders.

Assuming that combination of lenses in trial frame is — 1.00 D. Sph ⌢ + 2.00 D. Cyl. axis 90. When the cross cylinder is held with its axis corresponding to the cylinder of the combination and vision is improved, the sphere is increased ¼ diopter, and the cylinder is increased ½ diopter. If the improvement is noted when the cross cylinder is held at right angles to the axis of the cylinder in the trial frame, the sphere is reduced ¼ diopter and the cylinder is decreased ½ D. To summarize the action of minus spheres when used in conjunction with plus cylinders, together with the cross cylinder, let me say:

(a) When the cross cylinder is held with its axis corresponding with the axis of the cylinder of the combination, both the sphere and the cylinder powers are increased.

(b) When the cross cylinder is held with its axis at right angles to the axis of the cylinder of the combination, both the sphere and cylinder powers are decreased.

It will be noted in every case that the spherical power is affected to the amount of the sphere of the cross cylinder combination, and that the cylinder is affected to the amount of the cylinder of the combination.

Recapitulation of the Actions of the Various Lenses and Combination of Lenses When Used in Conjunction with Cross Cylinders.

(a) When used with spheres the sphere is reduced and a cylinder is created.

(b) When used with convex cylinders at (1) the cross cylinder used with corresponding axis, a minus sphere is created, and the power of the cylinder is increased. (2) When used with axis at right angles, a plus sphere is created and the power of the cylinder is increased.

(c) When used with plus combined with plus combination: (1) with axis coresponding, the sphere is reduced and the power of the cylinder is increased; (2) with axis at right angles, the sphere is increased and the power of the cylinder is reduced.

(d) When used with minus combined with minus combinations: (1) with axis of cross cylinder corresponding, the sphere is increased and the power of the cylinder is reduced; (2) with axis at right angles, the sphere is reduced and the power of the cylinder is increased.

(e) When used with minus spheres combined with plus cylinders: (1) with axis corresponding, both sphere and cylinder powers are increased; (2) with axis held at right angles, both spheres and cylinder powers are decreased.

(f) When used with plus on minus combinations, in which the power of the sphere is greater than the power of the cylinder, the action of the cross cylinder is the reverse of the plus on plus action.

(g) When used with plus on minus combinations, in which the cylinder is greater than the sphere, the action is the reverse of minus on plus combinations.

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A FEW NOTES ON VISUAL EFFICIENCY

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Within our time and memory we have seen the thought of, and pursuit of, maximum acuity give way to the bigger ideas of comfort, efficiency and safety. Optometry's motive, very properly, is to prevent injury to, restore and conserve, human vision in order to gain the manifest and manifold benefits that can flow only therefrom.

I believe more serious questions are going to be asked of the optometrist than how much can the patient see at a given instant and what improvement can be made in a particular case. The questions I have in mind are: how efficient is a given pair of eyes? how efficient is a single eye? at the present time? and under certain conditions? is the efficiency of a stable quality? will it rise or fall under probable circumstances and conditions? what are the conditions that will make for and against a probable rise or fall?

Those who think of optometry's work only in terms of maximum acuity will admit that one can have maximum acuity with a limited field; with enormous errors of refraction and muscle imbalances; with color failure, with slow associated communications; without stereopsis or depth perception; without pupillary function for necessary light control; with limited close-range function; and with attendant pathology.

Of what interest or value, it may be asked, can an efficiency rating possess? To the individual who has a present efficiency rating of fifty per cent, and knows it, though he possess a restored acuity of so-called normal or nearly normal, should be on the alert to build to a higher rating, if possible, and to take more than average precautions in the best interests of his ocular and general safety. To the examiner, it is an excellent guide to advise in a sound manner, and to observe positive or negative progress in time. To third persons, it is a guide and guard against personal injury, property loss, and reduced efficiency in employment.

It should not be too difficult to visualize a person having 20/20 vision, (apparent one hundred per cent acuity), having a visual efficiency rating of less than fifty per cent; or a person having 20/40 or less vision (apparent fifty per cent of normal acuity) having an actual visual efficiency rating of eighty per cent. I think it is not saying too much that it would be safer to ride with one having an acuity of 20/100, with other functions nearly normal, than behind

one having maximum acuity, with other functions sharply below normal.

Of common occurence are one-eyed people. Yet, I doubt if any one engaged in this visual science could answer at short notice the question "what is the visual efficiency of a one-eyed person having normal acuity in the surviving eye?" Such people are everywhere engaged in work hazardous to themselves and hazardous to others. You and I know that to be a fact.

Reducing to acuity percentages, giving a vague, misleading idea of efficiency, becomes manifestly absurd when one considers an infinite variety and range of errors or ocular perversions resulting in about the same lack of acuity in respect to distance. The presbyope and the myope have reversed areas of utility, the former lacking "near" utility, the latter "far" utility—yet, the acuity percentage favors the presbyope, whereas, in fact, in our present-day world of close adaptation necessity, the myope may have an efficiency for most purposes greater than an emmetrope. Furthermore, we are often misled as to the state of acuity by those minds who "see" by context, giving a picture in gross minus important details therein. Such minds may easily "read" the 20/40 letter, since the opportunity for context reading is afforded by an exposed area four times as great as the surface area at half the distance from the observer. It should not be forgotten, too, in many cases of ametropia or anisametropia, zones of fusion and maximum acuity are quite limited for binocular purposes. Too, in ordinary corrections with lenses, and faulty selection of frames, the field of vision may be materially reduced. Yet, such cases, possessing normal acuity centrally, ought to be shown as having a materially reduced visual efficiency.

One of the difficulties encountered at the outset, in any attempt to evaluate the factors on which visual efficiency shall be determined, is the overlapping of one or more of these factors. It must be evident, too, that there must be a difference in judgment of average values. Also, provision must be made for temporary and permanent ratings in respect to general efficiency and occupational efficiency.

In respect to errors of judgment on the part of the examiner, whereas the error constant in scientific *observation* is, I think, half of one per cent, the error constant of judgment must be much greater, for it is not a matter of simple comparison, but rests rather upon a complexity of abstract performances and extent of personal knowledge. Hence, I think five per cent is not too much, and it may be too little, so that, at a maximum, an individual may not have more than ninety-five per cent as maximum visual efficiency, which certainly is in order with the truism that we have no perfect thing or condition.

Now, the factors, in addition to acuity, that may be clinically dealt with in estimating visual efficiency may be said to consists of:

1. Peripheral acuity, or extent of fields.
2. Speed of vision.
3. Binocular value.
4. Stereopsis.
5. Light sensitivity (threshold and maximum).
6. State of accommodation.
7. State of extra-ocular muscles.
8. Color perception.
9. Lens-eye system (corrected).
10. Pathological sequalae or state of health.
11. Presbyopia.
12. Error of judgment constant.

Reflection leads me to believe that no single individual can so well prepare tables of Depreciation Values for the factors enumerated as can be arrived at in the judgment and experience of many optometrists throughout the world. I offer the suggestion, therefore, that each Academy of Optometry group, or its equivalent, work on these tables, submit their findings to a central board of optometrists and other qualified and interested scientists, who shall make final determinations of the mass findings as standards for the determination of Visual Efficiency. Undoubtedly, these standards should be examined at periodic intervals for refinement to perfection.

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OCULAR ELECTROTHERAPY

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CHAPTER VI

THERAPEUTIC ELECTRIC CURRENTS

There are several types of therapeutic electric currents which vary considerably in their therapeutic values. Therapeutic currents may be generally divided into two main classes:

1. High Frequency Currents.
2. Low Voltage Currents.

There are three distinct types of high frequency currents, which are produced by the different kinds of coils. The current produced by each coil has a separate and distinct characteristic.

The three types of high frequency currents are:

1. D'Arsonval
2. Tesla
3. Odin

The D'Arsonval current is one derived from a D'Arsonval solenoid (which will be described in this chapter) which is bi-terminal; of high frequency, high amperage and low voltage.

The Tesla current is of the highest voltage and highest frequency current possible to obtain from a diathermy machine having a Tesla coil incorporated in it.

The Odin current is of the highest voltage and highest frequency possible to obtain from a diathermy machine in which is incorporated an Odin coil.

The question may be asked "what difference is there between a Tesla and Odin current?" The difference is this, the diathermy current is put through a step-up transformer which raises the voltage and lowers the milliamperage leaving the frequency of the diathermy current the same as it was in the original diathermy circuit before it passed through the Tesla or Odin coils.

In our description of the production of high frequency current in the previous chapter it was stated that the first step in producing high frequency current is to raise the voltage of an ordinary alternating commercial current from 110 volts by putting it through a voltage step-up transformer. The frequency of this current, as it comes through the transformer, remains the same as it was before the current entered it. The next step is to take this current as it comes through the voltage step-up transformer and pass it through a circuit

consisting of a spark gap, condenser and a coil, and thus raise the frequency. The type of high frequency current it will produce depends largely on the type of coil, whether it be a D'Arsonval, Tesla or Odin.

The D'Arsonval coil is made up of a few turns of wire, which is connected up to the battery of condensers and spark gap. This hookup produces a current of *high frequency, high amperage* and *low voltage,* and is known as the *D'Arsonval current* or the *Diathermy current.*

The Tesla coil is made up of a tube of about two and one-half inches in diameter and fourteen inches in length wound with magnet wire, which is well insulated and this forms the secondary of the transformer. The primary consists of four turns of heavy, high tension auto-cable wound over the secondary. The turns of the primary are from 10 to 14 inches in diameter and the secondary is in the center of the large turns. The primary is a separate circuit and insulated from the secondary circuit on all sides evenly by several inches of air, as shown in figure 9.

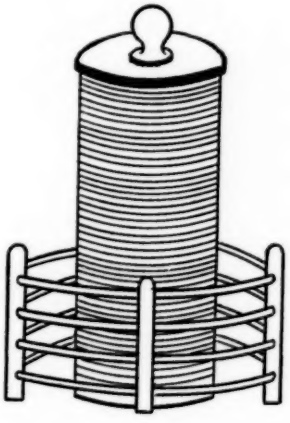

FIG. 9

Sketch of the Tesla coil (C. M. Sampson). The larger four outside turns of wire make up the primary coil and the numerous smaller turns on the inside make up the secondary coil.

Sampson in explaining the action of the Tesla claims that the current is generated in the secondary by the magnetic lines of force reaching out through the insulation of the primary and cutting through the turns of wire on the secondary. These lines of force start from the primary as the voltage surge in the primary starts to rise and have to travel several inches before they strike the secondary. As the voltage surges, recede or fall in the primary, these lines of force return to the primary, leaving the secondary

several inches before they return to the primary. There is a considerable interval of time both on the rise and the fall of the voltage when the secondary is not being acted upon at all by the primary changes. The result is that every cycle in the primary which has voltage enough to reach out to the secondary produces an abrupt rise and fall in the voltage in the secondary of very much higher voltage and of very much shorter duration than the voltage surge in the primary. We thus have the fundamental frequency of the circuit change by successive bursts of peak voltages with long intervals between each burst, and *the tissues react to these bursts exactly as they do to the slower bursts in the diathermy* circuit when these are slowed up by opening the spark gap too wide.

The Odin resonator is so constructed that both the primary and secondary are wound like a large coil clock spring[25], that is concentric and in one plane. The primary and secondary are connected as one continuous piece of wire and with consecutive turns almost touching one another, with only the slightest of insulation between successive turns, as shown in figure 10.

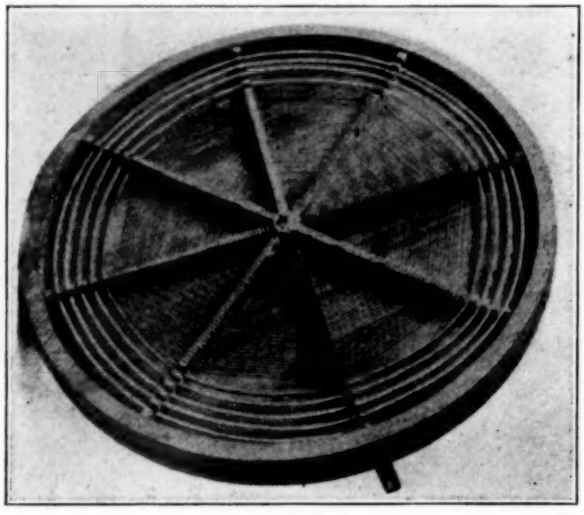

FIG. 10

Diagram of the Odin resonator (C. M. Sampson). The larger outside turns represent the primary coil, and the smaller inside turns represent the secondary. The box is filled with an insulating compound and is covered before it is placed in the diathermy machine.

The result here is entirely different than in the Tesla coil. Because nearly at the same instant that the voltage surge starts at the primary, the lines of force cut through closely the adjoined turns of the secondary and a voltage rise starts in the secondary and lasts until the voltage in the primary has returned almost to the zero

point. Thus, in the Odin the voltage rise in the secondary occupies nearly as long a time as in the primary and there is a very much less abrupt rise and fall of the voltage than there would be in a Tesla coil.

The Odin current due to its comparatively slow rise and fall of voltage is more tolerated and pleasant than the Tesla.

The D'Arsonval, Tesla and Odin currents may be differentiated as follows:

D'Arsonval current is one of high frequency, high amperage and low voltage. It is known as the diathermy current.

Tesla current is one of high frequency, high voltage and low amperage, and is not a very smooth current, it is farodic.

Odin current is one of high frequency, high voltage and low amperage, and is a smooth and pleasing current.

With the recent development in the manufacture of high frequency apparatus, various kinds of hook-ups were devised entirely different from the original coils of D'Arsonval, Tesla and Odin. The naming of the currents after the discoverers should be discontinued in view of the fact that the coils employed at present do not resemble the original ones brought out by the three mentioned inventors.

Currents of *tension* have reference to *voltage;* currents of *quantity* have reference to *amperage,* and *mono-terminal* currents of *high tension,* to their exceedingly high voltage and correspondingly low amperage, yielding what is called "cold spark."

High Frequency and Diathermy

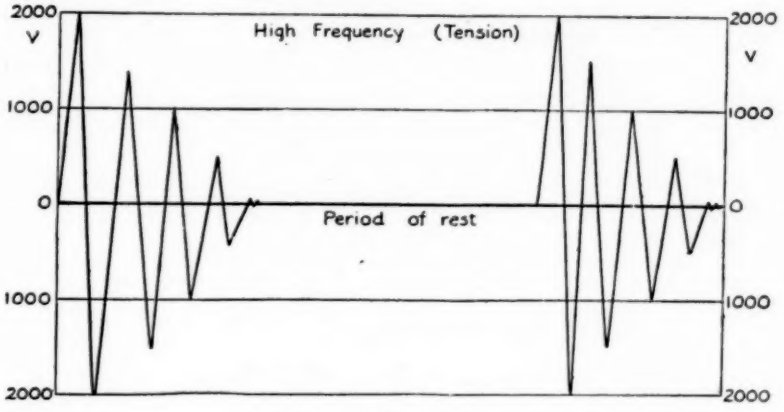

FIG. 11

Diagram of high-frequency oscillations of tension (Turrell).

Turrell[26] in discussing therapeutic currents maintains that there is a real physical difference between mono-terminal high frequency and bi-terminal diathermy currents. The former he claims has a

higher voltage (higher tension) and lower amperage (lower intensity) and the latter has a lower voltage (lower tension) and a higher amperage(higher intensity) and he suggests that instead of distinguishing between high frequency and diathermy currents, we should call the former High Frequency Current of Tension, and the latter High Frequency Current of Quantity. Turrell insists that the physiological and therapeutic difference of the two currents is a real one.

The physical difference of these two currents is illustrated by figures 11 and 12.

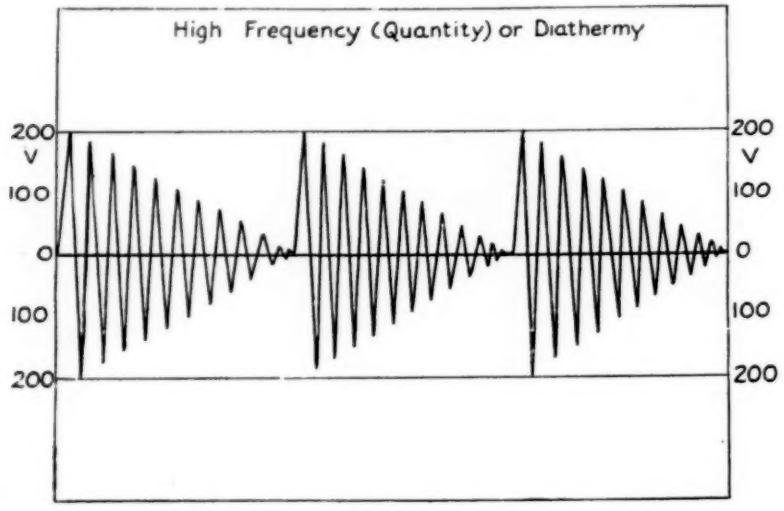

FIG. 12
Diagram of high-frequency oscillations of quantity-"diathermy" (Turrell).

Grover[27] gives the characteristics of some of the therepeutic currents and their classification as outlined in the table which follows:

CHARACTERISTICS OF HIGH TENSION, QUANTITY AND HIGH TENSION MONO-TERMINAL CURRENTS

	Voltage	Amperage	Oscillations	Spark	Effects	Frequency
High Tension	High	Low	Spaced	Hot	Stimulating	Varies in accordance with capacity, inductance and resistance of the circuit
Quantity	Low	High	Non-spaced	Flame	Sedative	
High Tension Mono-terminal	Very High	Very Low	Spaced	Cold	Deeply Sedative	

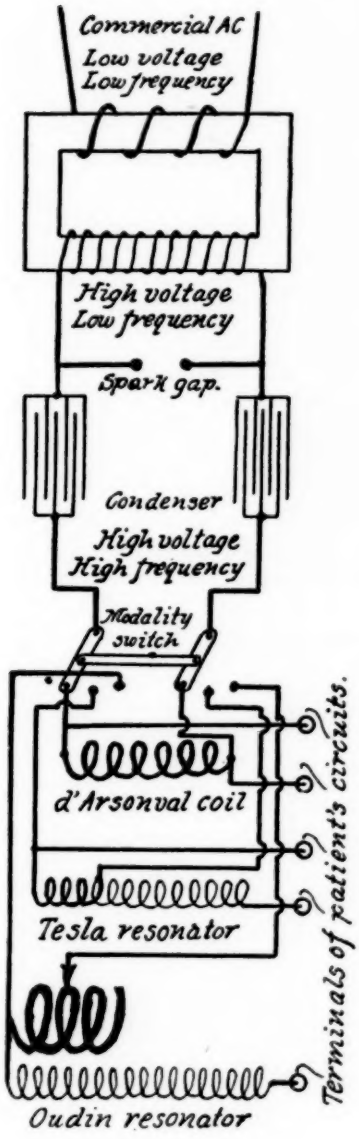

FIG. 13

Diagram of high-frequency hook-up which furnishes all three types of current, the d'Arsonval, Tesla and Odin.

Measuring High Frequency Current

High frequency current can not be measured by the ordinary electric meter, hence, a device called the hot wire is employed. This meter is introduced into the circuit and registers the amount of heat instead of strength of the current passing. Only the numbers of the milliamperes of current passing can be registered on this meter. This meter is really a thermostat instead of an electric device. It it based upon the fact that the passage of high frequency current gives rise to heat and that this heat causes expansion of the platinum wire and thereby deflects the needle in proportion to the amount of heat produced by the passage of the current.

This and the preceding chapter has been devoted to the description of the essentials of a high frequency machine. Upon these principles all machines are constructed.

Studying figure 13 one may get a graphic picture of the hook-up which supplies the three forms of high frequency current.

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REPORT OF THE TRANSACTIONS OF THE AMERICAN ACADEMY OF OPTOMETRY

Tuesday, December 6th, 1927, the American Academy of Optometry reconvened at the Hotel Pennsylvania, the Chairman, Dr. E. G. Wiseman, occupying the chair. Dr. Wiseman introduced Dr. R. M. Peckham.

VISUAL NERVE TRACTS[1]

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Another auxiliary pathway is through the 7th nuclei, a protective pathway. Through the 7th, by connector fibers from the 7th to the 6th, eye movements, facial movements, winking, are brought into association. A similar pathway extends from the hearing nuclei of the 8th, turning the eyes in response to sound. Most complicated of all, highly essential to our physical welfare, are the pathways from the equilibration system to the oculo-rotary nuclei and the proprioceptive pathways from the ocular muscles to the equilibration areas, by which eye movements are co-ordinated with body and head movement, visual impressions with equilibration sensations. This is a most interesting study, for it involves some of the hyperphorias and cyclophorias we find. Also, a very important matter with us, lack of synchronism of visual sensation with equilibration sensation is the cause of many of the cases of dizziness and car sickness of which some of our patients complain. In these cases, we find versions slow, ductions poor, tensions of muscles, sometimes the reverse, low tonicity. All of these are matters within our realm of practice and we need to know far more concerning these fiber tracts. This phase is too large to enter into at this time, it needs and deserves, a period devoted exclusively to its study.

So much for the versions. Now, when it comes to placing and maintaining both foveas beneath both images, we need a more complicated system of pathways. Not much is known, unfortunately, about these. There have been recognized fiber paths from the right voluntary 3rd nucleus to the left 3rd nerve, fibers from the voluntary left 3rd nucleus to the right 3rd, and it is thought that through these are sent the impulses needed for this associated foveal adjustment. Imagine our horseman, who pulls both horses to the right with his right hand and both to the left with his left hand, holding an extra pair of reins in his hands by which he could pull his horses toward each other. Then perhaps you picture, in a very rough way, the use of this newly developed pathway, I say "newly developed," for it is

[1]Continued from May Issue.

only present in the higher mammals and is not well developed save in a few of the apes and in man.

Now for the accommodation pathways. They are not definitely proved. As skill in dissection advances, we have to change our ideas overnight.

From the retina, the fibers go to the optic tracts. Here they end, discharge their impulses. From here they are relayed to the external geniculate bodies. Some say there is also a relay pathway to the pulvinars of the thalami. Some deny this. There are pathways from the optic tracts to the pregemina. It was long thought that there were relay pathways from the pulvinars to the pregemina. But this is now doubted. I had fourteen dissections made by careful and competent technicians. None of the fibers said in the anatomies to pass from the pulvinar tracts to the pregemina were found.

For a long time it was thought that sensations leading to muscular actions were sent through the external geniculate bodies and that sensations of form and color were sent through the pulvinar. This is now denied. The external geniculates lie on the outer walls of the thalami. The pulvinars are the posterior portion of the thalami. It is now thought that not only the sensations causing muscular movements but also those of form and color are relayed from the optic tracts to the external geniculates. There are fibers connecting the external geniculates with the pulvinars. There are also fibers connecting the two external geniculates. There are fibers connecting the two pulvinars.

The fibers from the retinae end in the optic tracts in the same relative position as their outer terminals in the retinae occupy. The same relative position is carried into the external geniculate bodies. In reality, the external geniculate bodies are continuations of the optic tracts.

Histologically, the optic tracts and optic nerves resemble the gray matter of the brain. They do not appear, under the microscope, like the gray matter of true nerves.

From the external geniculate bodies and from the pulvinars are heavy masses of fibers leading to the occipital lobes of the cortex and to the frontal lobes.

The occipital lobes have always been considered as the seat of visual impressions. Lately, some physiologists have advanced the theory that some visual impressions are conveyed to the frontal lobes, but nothing tangible has been brought forward in proof. We shall have to continue with our thought that the visual centers lie entirely in the occipital lobes.

Accommodation impulses pass through the pregemina. (The word pregemina is a recent abbreviation for the long term "Superior Colliculi of the Corpora Quadragemina.) The question is: Is accommodation incited over the pathway from the External Geniculates

or must the sensory impulse first pass to the cortex of the brain there to be transformed into a motor impulse, sent to the pregemina to be relayed on through various nuclei to the ciliary muscles? There are very conflicting opinions as to this.

Primitive impulses are completed through the bodies of the brain stem and spinal cord without reference to the cortex. Only impulses requiring mental attention go through the cortex. Is accommodation that type of primitive impulse that can be completed, in response to retinal blur, without direct mental attention?

The muscles of accommodation in the bird are voluntary muscles. In mammals, the muscles of accommodation are involuntary muscles, that is, like the muscles of heart, stomach, intestines, glands, etc., they are operated without mental attention. They are autonomous.

Is accommodation an innate function? There is dispute as to this. Some say yes, others say no. Do we accommodate, can we accommodate, when we are born or must we wait until the nerve pathways are developed, a few weeks after birth. No one knows.

Sensory nerves are gray matter covered with a white sheath. The connector nerves of the brain are not covered with this white sheath or myelination, until the individual is ready to use them. The nerves from the external geniculates to the occipital lobes do not take on their white sheaths until the baby is three weeks old. That is when "He begins to see." That means, his consciousness has so evolved that he is ready to use his eyesight. Now if accommodation is not an innate function, and if accommodation requires cortical attention, then the pathway from the external geniculates to the cortex are essential to accommodation. If these pathways are not essential, if accommodation is a primitive function, if its reflex arc is completed through the pathway from external geniculates to pregemina, then the cortical pathway is not necessary. I proposed to prove this one way or the other by cutting the fibers from external geniculate to cortex, in a monkey, permitting him to recover, and then test his accommodation. The first attempt with the experiment was a failure and I have not had opportunity for another trial.

At any rate, we do know that the impulse passes through the pregemina. From here there is another question, not satisfactorily proved, to which I will return in a moment.

The next relay is the involuntary part of the 3rd nucleus. It seems very odd that the students in all but one of our schools have never been taught that the 3rd nucleus and the 3rd nerve are in fact three distinct nuclei and three distinct nerves, not at all associated, and having no influence one upon the other. Had this been given the attention it should have received, most of the things that have been written about associations between convergence and accommodation would never have been uttered.

The involuntary 3rd consists of two parts, the nuclei of Edinger Westphal and the mesial nucleus. From the pregemina, fibers go to

the nuclei of Edinger-Westphal, thence to the mesial nucleus, then to the ciliary ganglia, then into the fibers of the ciliary muscle.

Now here is another question awaiting solution. The ciliary muscle is in fact three distinct muscles. There is a bundle of circular fibers, surrounding the lens, imbedded in the ciliary processes. When they contract, the ciliary body is pulled forward, toward the lens, the suspensory ligaments are relaxed, the lens becomes more convex. That these receive their innervation through the involuntary 3rd is well enough established.

Antagonizing these are the radial fibers, which in contracting pull back the edges of the ciliary body, tighten the suspensory ligaments, flatten the lens. Whence do they receive their innervation. No one knows.

Then there are the meridional fibers, attached at one end to the sclera at the base of the iris, inserted at the other end into the choroid. When they contract, they pull the choroid forward, the ciliary body moves forward, the lens becomes convex. Whence comes their innervation. Clinical evidence indicates that it comes through the spinal sympathetic system. Anatomical evidence supports, to some extent, this view, though the full path of the fibers has not been definitely mapped out. Physiological evidence is lacking.

From the pregemina, besides the fiber tracts to the involuntary 3rd, there are other bundles that pass down the brain stem to the cervical segments of the spinal cord, where they discharge in an area known as the cilio-spinal center of Buge. Here the impulses are picked up by another relay of fibers and carried into the superior cervical ganglia. Thence emerge fibers to the intrinsic muscles of the eye.

It is known that this pathway just described carries the impulses that dilate the pupils. It is thought that they carry impulses to the meridional fibers and the radial fibers of the ciliary.

The superior cervical ganglia are at the head of two long chains of sympathetic system ganglia, connected with and innervating the involuntary muscles of the skin and internal organs of the body. This is known as the spinal sympathetic system.

All muscles have antagonists, either other muscles or elastic tissue. In the involuntary system, one set of muscles is innervated by the spinal sympathetic system, the opponents by the para-sympathetic system.

The sphincters of the iris are innervated by the para-sympathetic system, the dilators, by the spinal sympathetic system. The involuntary 3rd is a part of the para-sympathetic system.

When any of the vital organs is so affected that extra effort is demanded from either para-sympathetic or spinal sympathetic centers, the influence spreads throughout the entire system, all organs are more or less affected. When one system is over active, the oppo-

nent is proportionately inhibited. Tonicity of the muscle that is put to extra work is increased, a corresponding amount of tonicity is withdrawn from the antagonist.

When there is a disturbance of the digestive organs or of the sexual organs, such that the spinal sympathetic system is called on for extra duty, we get a reflex in the eyes by a dilatation of the pupils. Or if the para-sympathetic system is overworked, we have contracted pupils.

In the myopic eye, on dissection, we find the meridional fibers very heavily developed, the circular fibers are few and small. When a muscle is used it develops in size and in tonicity. When it is unused, it grows smaller, its fibers waste away from lack of tonicity, lack of use. The nerve fibers to it degenerate.

I have several slides made from known myopic eyes. In all of them, the meridional fibers are heavy; under the microscope they look like great cables. In one of them, there are no circular fibers to be found, and but slight traces of radial fibers. In the others there are a few tiny circular fibers, a few weakly developed radial fibers.

The most advanced thought is that myopia is a symptom of visceral imbalance, that the reflexes have spread throughout the spinal sympathetic system, contracting the meridional fibers of the ciliary, putting them into a state of tetanus, what has been falsely called "Spasm of the Accommodation." Likewise, the pupils are highly dilated, as we can see without a microscope. On dissection, the dilatators of the iris are seen to be heavily developed, the sphincters very slight, evidently weakened, since all tonic and trophic innervations have been withheld.

On the other hand, we find the circular fibers of the known hyperopic eye to be heavily developed and the radial fibers, their antagonists, are very few in number. The meridional fibers are also underdeveloped.

Several physiologists have experimented with spinal sympathetic innervation of the ciliary, with negative results. They have learned nothing. But they are decidedly wrong in therefore assuming that there is no such innervation. Their experiments were not sufficiently thorough, probably they lacked equipment, doubtless they needed more knowledge. We are not to consider the problem as settled. No better undertaking could be assumed by optometrists than the attempt to solve this question. It would mean years of work by skilled laboratory workers, the expenditure of thousands of dollars. But it is a question that must be answered, and to the optometrist it is of particular importance. For therein lies the answer to the vexatious contention that atropine is the only way of determining the amount of latent hyperopia.

Atropine only paralyzes the para-sympathetic nerves. Only the action of the circular fibers of the ciliary is abrogated. Further, when the para-sympathetics are rendered non-conductive by atropine, their

normal tonicity is discharged into the spinal sympathetics. That is why the pupils dilate so rapidly, why the heart beats faster and irregularly, why the face flushes.

Now we can conceive of two reasons why we can often find more latent hyperopia after three or four days' use of atropine. One is that the radial fibers, in a highly hyperopic eye, may be slight and weak. Possibly, if it is true that they are innervated by the spinal sympathetics, they cannot receive the full tonic discharge that is switched from the para-sympathetic system under inhibition. If that could be possible, then we could see that the para-sympathetic would not be fully inhibited. That is an experiment I hope to make some day. Another conceivable supposition is that the excess tonicity is sent into the meridional fibers, causing them to contract in the same manner that the dilatators of the eye are forced into excessive flexion. Neither of these suppositions, and that is all they are, can be denied or affirmed, until the question of spinal sympathetic innervation to the ciliary is proved or disproved, finally and without doubt.

Dr. Savage made a noteworthy statement at the 1927 Ophthalmological Congress. He said that no known drug would paralyze Bowman's muscle. Bowman's muscle is the old name for the meridional and radial bundles of ciliary muscle fibers. Such a statement from such an active protagonist of the use of atropine as Dr. Savage has always been is worth attention.

We even have records on file of myopic children being carefully measured as to the amount of myopia, then put under the influence of atropine for three days and becoming more myopic. Surely, all these things mean something. Let us find out the truth, if it be possible.

Nicotine is the only known drug that will render a spinal sympathetic nerve non-conductive. And it has to be applied to the naked axis of the nerve as it emerges from the spinal cord. Not a usable method in routine refraction work.

A word as to the pathway of pupillary contraction. Fibers are found paralleling the accommodative route fibers from the third nucleus to the eyeball. These are known to carry both light reflexes and the associated contraction that accompanies convergence-accommodation impulses. Possibly there are two sets of fibers for these two different tasks. And the associated convergence-accommodation pupillary contraction has been satisfactorily traced from pregemina to third nucleus. But the light reflex pathway is still open to investigation. How it gets from the optic tracts to the third nucleus has not been assuredly determined. It has always been stated that the pathway is through the external geniculates and pregemina. But now that whole theory has been shown to be entirely speculative by some German physiologists. Several researchers have shown that the sphincters of the iris retain the qualities of primitive muscle, which reacts from contact without the interposition of a nervous system. After

cutting both the sensory nerves from the sphincters to the central system and the sensory nerves from the retina to the optic tracts, also severing the motor nerves to the sphincters from the central system, they found that the iris would contract to bright light. So far as is known, this is the only muscle in the human body that still possesses the autonomous contractility of the muscles of primitive invertebrates.

Having covered, somewhat sketchily, but as well as time will permit, the pathways of the versions, of independent convergence, of accommodation, of pupillary control, we can revert to the convergence-accommodation pathway. It is thought that the sensations discharged in the occipital lobes are transmitted over association pathways, to the parietal lobes. Here associative memory plays its parts. Old impressions are reawakened, the picture is compared with sensations received in the past by way of touch and pressure sensations, heat sensations, auditory impressions, word pictures, etc. The whole is fused into a new picture, recognized for what it is. The upper part of the parietal lobe has been shown to be an associative field for the information we gain from the various sensations.

Further down is the field of word forming. Near this is the motor field that arouses the convergence-accommodation function into activity when the image received in the retina is out of focus, when the "picture," for we have no better word, is not clearly delineated to this unknown observing function that we call Mind.

From here the fiber pathways seem to pass forward to the pregemina. And here they terminate, form new bundles and pass to the oculo-rotary nuclei. Now enters the most astonishing thing of all, when we consider what we have been taught in the past. If this be finally proved true, then optometric procedure is subject to considerable modification, on its theoretical basis, at least, if not so much in its practical application.

The evidence, at present, points to a simultaneous innervation of the 4th nuclei, which supply the superior obliques, and the involuntary division of the 3rd, which supplies the circular fibers of the ciliary and the sphincters of the iris. And the voluntary section of the 3rd, controlling the internal recti seem to be left out.

However, we must not go too fast nor jump to conclusions too readily. Without doubt, there are other associative pathways, not yet discovered, that bring the internal recti and inferior recti into the association. But this much we may be fairly safe in accepting, that there is no known pathway by which the internal recti are brought into associated activity with the function of accommodation.

There is another point to be remembered. Cortical innervations, that is, motor impulses from the cortex, are never, in any part of the body, sent to the muscles that are to be used in the willed act. The cortical impulses are sent into nuclear areas, where they are sorted and arranged, directed to the specific muscle or muscles

that are to be used. The action of the muscle and its association with other muscles is arranged by reciprocal innervational pathways interconnecting the nuclei of the nerves that supply the muscles.

We have just enough information now about these associations of extrinsic and intrinsic muscles to make us seriously question our classics, enough to show that our writers have been speculative in the extremist of extremes, enough to awaken our curiosity and start us out into real investigative work, determined to discard the speculative, to seek for facts.

The 6th nuclei and the voluntary divisions of the 3rd when innervated in the willed acts of version and convergence receive the impulses through the medial longitudinal fasciculus, which arises from two pairs of nuclei in the roof of the midbrain. These are the interstitial nuclei of Cajal, which lies in floor of the third ventricle, and the nuclei of Drakschewitsch which lie in the upper and forward sides of the pregemina. It is thought that the impulses leading to eye rotations are sent from the oculo-motor fields of the frontal lobes through the nuclei of Drakschewitsch. The right 6th receives fibers from the left bundle of the medial longitudinal fasciculus, the left 6th receives from the right bundle.

Both right and 3rd nuclei receive fibers from both right and left bundles of the medial longitudinal fasciculus. The right and left nuclei of Drakschewitsch are connected by a commissural bundle of association fibers, running through the posterior commissure of the brain stem. Thus, by this commissural bundle and the semi-decussation of the fibers from the medial longitudinal fasciculus, the 3rd nuclei are brought into that integrated association demanded in the complex movements of lateral, vertical or oblique rotations with maintenance of convergence of the visual axes, so that the foveas will remain beneath the two images throughout these ocular excursions, constant single vision will obtain.

By the pathways of control from each 6th to the opposite 3rd, reciprocal innervations and inhibitions, essential to balanced binocular movements, are enforced. The control of the 4th nuclei from the 6th is a part of this system. The whole presents one of the most beautiful pictures of co-ordinative pathways to be found in the body.

The 4th is innervated from the visual motor nuclei in the pregemina. These fibers pass downward from the pregemina through the medial longitudinal fasciculus to the 4th nuclei. For a long time it was thought that these fibers were functioned within the medial longitudinal fasciculus and they are so given in many anatomies. Recent tracings show these fibers to the 4th are not a part of the system just described as feeding the 3rd and 6th. The 4th seem to be excluded from direct control through the medial longitudinal fasciculus while the 6th and voluntary divisions of the 3rd do not appear to be controlled from the pregemina. The fibers from the pregemina to the 3rd are evidently all involuntary fibers to the involuntary nuclei of Edinger-Westphal.

This is an important point. For here is a part of the evidence of the associated innervation of the superior obliques with the circular fibers of the ciliary and the sphincters of the iris in the functionation of convergence-accommodation. It is also part of the evidence that makes us doubt the direct association of the internal recti in the act of convergence-accommodation.

Then we run into another block that gives us pause. For there is no agreement as to the rotation of the eyeball that is caused by the flexion of the superior oblique. The old anatomies give this as an out and down rotation, an error still persisting in some of the anatomies and in most of text books. A later idea has been promulgated by some ophthalmologists that the rotations produced the superior obliques are in and down. Now a third theory is presented by the neurologists, who say that the rotations are in and up.

Touch the right pregeminum of the monkey with an electrode and the left eye rolls in and up. Touch the left pregeminum and the right eye turns in and up. Touch both pregemina and both eyes turn in and up and the entire body begins to jerk and quiver. I have performed this experiment, which is described in several books on nervous diseases, and the results are as stated.

But the act of convergence-accommodation carries with it an in and down rotation of the eyeballs. So there is still something to be learned about these innervational pathways. Either the neurologists are wrong and the ophthalmologists are right about the action of the superior obliques in the human, or there is an undiscovered pathway bringing the inferior recti into association. This is another problem that might be undertaken by a research organization of optometrists.

The neurologists assert that an in and up squint is a symptom of disturbance in the pregeminal pathways. That the superior oblique is in a state of tetanus as the result of a spasm, or convulsion. When the pregemina are affected, or when one of them is affected, by diseased conditions, there is a noticeable interference with ocular rotations. Accommodation and pupillary contraction are nearly, sometimes totally, suspended.

In squint of the in and up type we will note that invariably the pupils are dilated. With skiascopy we find an abnormally high lag of accommodation. This has been considered as an evidence of latent hyperopia. But this, I think, is a wrong conclusion. It is more probably an evidence of the partial suspension of the convergence-accommodation function. Plus lenses will not assist in the straightening of the deviating eye. In fact, the patient usually grows more cross-eyed when wearing the heavy plus found by dynamic skiascopy.

Until a very short time ago, in and up squint was considered incurable save by operation. But in the last year, two Academy members, considering the above data, have succeeded in straightening cross eyes of this type. Dr. G. M. Martin, of Utica, N. Y., and Dr. Frank H. Roberston, of Blackwell, Oklahoma, working independently,

neither knowing what the other was doing, have evolved a technique, identical with both, of straightening these eyes. The strong plus lenses are omitted, base-up prism over the squinting eye is given for constant wear, and by Kratometer treatments, bases-up over the squinting eye, the tetanus of the superior oblique is broken, the eye is brought to straight position. Convergence-accommodation begins to function and there is reduction of the plus seen in skiascopy.

The gratitude of the Academy, of all Optometry, is due to these two members.

And out and down squint, conversely, would indicate a paralysis of the pathway supplying the superior oblique. One would say, off hand, that such a squint would be incurable by prismatic treatment. However, an optometrist in Kansas, whose name at the moment I forget, showed me photographs of a case of out and down squint that he had corrected by Kratometer exercises. In this case, either there was not a total paralysis, some of the fibers in the pathway still retained conductivity, or the exercises had awakened such interest in the central system that another pathway was built.

We must remember two things. That nerves can be regenerated, do regenerate, not only after operations in which they have been cut if the operator carefully brings the cut ends together, but also following a diseased condition during which they become non-conductive or their nuclei become non-irritable. Or, failing this, the central system will often build another pathway, over some round-about route, to the muscle. The essential feature is that the central system shall be awakened to the necessity of such building. It seems fair to believe, with the evidence before us, that exercises may sometimes awaken such an interest.

All muscular activity, of whatever sort, is the result of disturbances in the central nervous system. We are thrown out of balance with environment by sensory messages to the central system. Our immediate act is the innervation of muscles so that we may be brought back to a state of content with our environment. Doubtless Professor Raup brought this out very clearly in his lecture on "Complacency." Personally, I consider his book on *"Complacency"* one of the most important contributions to optometry that has ever been made, though it was written with no thought or knowledge of optometry's peculiar problems. But in it are set forth fundamental conceptions of the physiological and psychological reflexes from which all human activity results. It is a book well worth careful perusal by our Academy members. Such study will show the errors into which many of theorists have been led.

Muscular activity is induced by dissatisfaction. The force of the muscular action, its quickness, energy, efficiency, is determined by the extent of the dissatisfaction, is proportioned by the interest excited. That must be the foundation on which we shall build our technique of innervational treatments.

Convergence-accommodation is an attention reflex. That statement cannot be reiterated too often. Its efficiency is based wholly on the interest awakened in the cortical centers, by the extent to which the blurred images create dissatisfaction. Given interest in the object, convergence-accommodation is enforced. When interest is lacking, when curiosity is not awakened, the function is not used to its fullest possibility.

In testing vision, I have been greatly surprised to learn that in many adults letter vision is poor, but given some other test object, something that awakens the curiosity of the subject, vision will sharpen up considerably. In testing the vision of small children, I have found that colored pictures awaken more interest, with consequent better visual records, than do the ordinary black drawings that we use on our test charts. I have also found that sometimes if the mother sits across the room and holds the test target that the child will give better answers than when the same target is shown in the test cabinet.

These are illustrations showing what I have attempted to bring out, that sharpness of vision depends much on the interest awakened. Attention must be incited, curiosity awakened, dissatisfaction with the blurred image must be present. Convergence-accommodation only results when there is discontent, "complacency" must be disturbed.

Here, I think, is the basic answer to the question as to why some persons are latent hyperopes while others do not make the effort to compensate for their structural hyperopia. These latter are not greatly disturbed by the blurred images, they are not sufficiently interested in the objects at which they are looking. The former are of a curious, investigative type of mind. They want to see. Therefore, they make the effort, they do see clearly, in spite of the possible strain involved.

Consider the non-presbyopic adult with 20/XXX or 20/XL, or possibly with worse, vision. He shows a manifest hyperopia, needs plus lenses, perhaps needs cylinders, to clearly see the distance test chart. But hand him fine print, a photograph or print, a piece of engraving with fine lines, anything to hold in his hand and look at closely, he will see the finest details. He will compensate his structural error and get clear vision. Why? Because his interest is awakened.

We do not measure "visual acuity" with our tests. It is wrong to use that term. What we measure is letter vision, and the determination of that measurement depends solely on the interest the subject has in those letters. His acuity may be good, it is, for he sees what he wants to see. We have as yet developed no technique for measuring "visual acuity," for we cannot separate the interest factor from the resolving power of the eye.

A few years ago I was making an optometric survey of the employees of a brass factory. One of the young women had 4.50 D.

of astigmatism in the right eye, 4.00 D. of astigmatism in the left eye. The axes were oblique. Vision at distance was about 10/CC by the letter charts. In the near tests with printed matter, she made horrible work of even large print. I inquired as to the work she was doing. Was amazed to find she was inspecting the brass ferrules of pencils. Of course, I went to the factory superintendent with the records, inquiring why a girl with such poor vision was doing such work as inspecting, which required the finest visual discrimination. Looking up the girl's records, we found that she had been an inspector for twelve years, was one of the fastest and most accurate of the inspectors. She compensated her structural error because she wanted to see her work. With printed matter, she did not compensate because she was not interested. With pictures I found her near vision was most excellent. With distant colored test objects, pictures that would awaken her interest, her vision was better than 20/LX, as nearly as I could estimate it. Compare that with the letter vision of 10/CC. Again, I repeat, convergence-accommodation is an attention reflex, a psychic matter, based on the interest awakened, proportioned to the dissatisfaction with the blurred image.

Take the child of eight or nine with 20/XX or better vision. Somewhat better than we normally expect at his age. You will find him a curious, interested, alert boy. If you make a thorough examination, you will probably find considerable latent hyperopia. You are apt to find a little esophoria at distance.

What is accommodative esophoria? Whence its origin? On dissection, we find that the convergence muscles are actually hypertrophied, are heavy, strong bundles of fibers. The anatomists of the last century, finding this, concluded that this was a congenital structure. Hence, the recommendation of operative measures.

Today, we doubt that this is a congenital malstructure. For muscles develop with use. They are kept constantly in a state of tonicity, so long as an exhaustion period is not set up. While in this state of high tonicity, their cells are rounded, the muscle is in consequence short and thick. When the exhaustion stage appears, the cells lose their roundness, tonicity is low, the muscle lengthens, is thinner. That is why we often find middle-aged men esophoric in the morning, exophoric in the evening. In the morning fresh, tonicity high. In the evening tired, tonicity low.

At the same time that we find the convergence muscles thus highly developed, in the known esophore, we also find the circular muscles of the ciliary equally overdeveloped. Why this development? Evidently because at an early age the individual learned to use his convergence-accommodation. The muscles are in consequence very highly developed. Their antagonists are correspondingly underdeveloped, sometimes partially atrophied.

Accommodative esophoria, therefore, is to be looked upon as a sign of the excessive use of the convergence-accommodation function. We know, from experience, that if we can correct the larger part

of the latent hyperopia, if we can persuade the patient to wear his full correction, that he will be fairly comfortable. That is, if he can tolerate blurred distance vision. After a time, some of these patients will learn that by not using their convergence-accommodation, they can have clear distant vision with the strong plus lenses. But such are few. The majority cannot adapt themselves.

The esophore's natural reaction to a blurred image is the use of convergence-accommodation. That trick was learned in early youth, has become a fixed habit. So we frequently find an esophoria increased by strong plus lenses, when those lenses blur the distance vision. Relaxation of the convergence-accommodation is unknown to the patient. His "set" is toward the positive act, the negation is impossible.

With base-in prisms we can enforce this relaxation. For such prisms deflect the images to the nasal sides of the foveas. The reflex act is the innervation of the 6th nuclei, to turn the visual axes outward. Innervation of the 6th means inhibition of the 4th and 3rd, through the associative pathways connecting these nuclei.

One point in connection with inhibition. Inhibition is only accomplished by innervation of the nuclei antagonistic to the nuclei we wish to inhibit. We do not inhibit a muscle. We inhibit the innervation of the nucleus of the nerve supplying the muscle. The only way to accomplish this inhibition is by innervation of the antagonistic nuclei.

So we inhibit convergence by innervating the 6th nuclei by base-in prisms that place the images on the nasal sides of the foveas. The reflex arc of an image on the nasal side of the fovea is the innervation of the 6th nuclei with reciprocal inhibition of the 4th and 3rd. Such prisms must be of equal power over both eyes. Prism over one eye only innervates one of the 6th pair, only inhibits one of the 3rd pair and one of the 4th pair.

The reciprocal inhibition that follows the innervation of a nucleus, has been found to extend far back into the pathway leading to the inhibited nucleus. That is, not only is the nucleus itself inhibited, but there is inhibition along the entire pathway, back to the original source of the innervation. Sherrington thought that the property of reciprocal innervations lay entirely in the nucleus. Later physiologists have found that reciprocal innervations are developed farther back in the pathway.

So with base-in prisms, with their innervation of the 6th, the antagonistic inhibition extends not only to the 4th and 3rd nuclei, but beyond them, with inhibition of the pregemina and convergence-accommodation, with inhibition even behind these to the cortical fields themselves.

Thus is it possible the complete abrogation of convergence-accommodation that we find when we use strong base-in prisms. For there is a point where, when we suspend convergence we also suspend

accommodation. Thus we can determine very closely the amount of latent hyperopia with base-in prisms, and we find, by comparative tests on the same people, that we can determine the latent hyperopia more accurately with base-in prisms than with atropine.

There are some theorists who have denied that this is possible. We can dismiss them with this, that in their writings they show very little acquaintance with the visual fiber tracts, very little acquaintance with physiological functions. Their denial is also based on speculative theory, for they admit that they have not given the method full experimental trial. And no one is in position to affirm or deny the value of any method or proposition until he has given it full and exhaustive trial.

The Maddox theory of over convergence by the esophore with following adjustment by the "faculty of supplemental divergence," is not found to be true. By laboratory methods, I have seen that the esophore does not over-converge and then come back to the point of fusion. He converges to the point of the single image, the visual axes do not pass their appointed intersection in the object of regard. The foveas rotate to the point of placement beneath the image and there halt.

It is only when we so displace the two images that only one fovea can be placed beneath its image at a time, as in the Landolt method of finding the horizontal phorias with base-down prism over one eye, or when we give to each hemisphere of the brain a different object to think about, as in the Maddox Rod test, that the visual axes converge too far. They do not over-converge during the act of binocular fixation.

Base-out prisms should never be given the esophore in so-called "position of correction," for such prisms actually force the over-convergence. They develop still more tonicity of the convergence muscles, the esophoria is increased.

Base-in prisms may well be given to all esophores. For such prisms inhibit the activity of the convergence muscles, withdraw tonicity impulses from them, divert that tonicity to the abducting muscles which have been in a state of lowered tonicity. We must always bear in mind, that when a muscle is made to contract, tonic impulses are sent into it and an equal amount of tonicity is withdrawn from the antagonist. If we can but remember that at every step, we shall not fall into erroneous prism prescription.

There is no such thing as "prism in position of rest." A prism always innervates the muscle beneath its apex, adds to its tonicity. The muscle beneath the prism base is always inhibited, tonicity withdrawn from it.

The use of the superior obliques in the convergence-accommodation and the control of the 6th nuclei over the 4th, gives us an understanding of the spurious hyperphoria so often found in cases of esophoria with latent hyperphoria. When we use our base-in inhibitory prisms and supply the needed plus correction, this hyperphoria

disappears. We find that this spurious hyperphoria is invariably a left hyperphoria in right-handed persons, a right hyperphoria in left-handed persons.

Myopia can be reduced, many times, by the use of base-in prisms. Or, if it cannot be reduced appreciably, vision can be greatly improved. Just why this improvement in vision occurs, I do not know. An ophthalmologist friend who has noted this suggests that it is because the withdrawal of tensions increases the blood circulation in the retina. That is a very plausible explanation, but even if it is true, we shall have to work farther back and find why the circulation is bettered.

Just as esophoria is to be regarded as a symptom of hypertonicity of the convergence muscles, so is exophoria to be looked upon as indicative of their lowered tonicity. Orthophoria is not to be considered as an indication of perfect muscular structure as the anatomists of the 19th century thought, for the normal muscular structure is such that the visual axes diverge widely. Orthophoria is an indication of tonic balance of the extrinsic muscles.

Emmetropia is hardly a condition of structural perfection for we find all emmetropes are structurally somewhat hyperopic. When we find emmetropia with orthophoria, there is always quite a degree of latent hyperopia.

The causes of exophoria are many and varied. Very few exophorias originate in the troubles of binocular functions. Most of them are symtoms of physiological, sometimes pathological, disturbances in other organs.

Comparison of distant and near phorias is of great value in diagnosis, especially if made before refractive errors are corrected. The phoria test made after correcting lenses have been supplied is of very little value unless compared with the phorias made at the beginning of the examination. Phoria tests are not "muscle" tests, they tell little about "strong" muscles and "weak" muscles. But they do tell us much concerning the habitual convergence-accommodation habit of the individual.

Duction tests do not measure "convergence reserves" or "fusional reserves." In fact, we gravely doubt if there is any such faculty as is indicated by those terms. They seem to have been coined in the need for representing such theoretical proposals based entirely on speculative thought, not on actual research, as we of today consider research.

Probably induction is poor in esophoria because the muscle is already in a state of hyper-tonicity, unable to accept an added load. Ductions may be low because of hyper-tensions, because of impaired nerve conductivity, because of a non-irritable condition of the muscle. The whole is a physiological problem and needs laboratory research to do away with the guessing of the past.

Negative relative convergence at the near point is a valuable aid in diagnosis, a feature that has not been given the consideration

it deserves.

When the negative relative convergence is low, we have a sure sign that the patient's complaint arises from an uncorrected latent hyperopia. With comfortable near vision, the negative relative convergence runs around 15Δ to 18Δ, depending somewhat on the interpupillary distance. It should equal, we find, the meter angle of convergence at the distance at which the test is made. When less than this, latent hyperopia is surely making trouble, must be determined and corrected.

Latent hyperopia lies at the root of many of the esophorias, exophorias, hyperphorias and cylcophorias that we find. The latent hyperopia must be determined in every case before we can safely proceed with our corrective methods. No examination is thorough, none is worthy the name "examination," that does not include a determination of the latent hyperopia and the negative relative convergence.

We should be very cautious about base-out prism treatments. They must not be given when the cause of the low induction is associated with latent hyperopia or with esophoria of any type. In those cases, base-in treatments are called for. We find that treatments with base-in prisms are the quickest and surest way of raising the induction in cases of hypertensions.

Base-out treatments may only be given when an exophoria arises from low body tonicity or when the low induction can surely be ascribed to low nerve conductivity or to non-irritability of the muscle. And then general bodily exercise should regularly accompany the prism treatments.

Another problem that confronts us is presented by the many cases of under developed functionation of the convergence-accommodation. These cases have been miscalled "premature presbyopes." The high plus movement found in skiascopy has been falsely labeled "latent hyperopia." This is in reality, I believe, a lag of the convergence-accommodation. Plus lenses are not needed, are wrongly prescribed. Base-out prisms will enforce some attention, awaken cortical innervation. Base-out treatments, using brightly colored pictures for targets, in particular red pictures, will incite the attention reflex needed.

The speaker went on to detail methods of examination procedure and suggestions for the treatment of various imbalances.

In conclusion he called attention to the need of a better understanding among all refractionists, both the optometrists and the ophthalmologists, of physiological principles. The problem of the refractionist has been considered as largely an optical problem; he has concerned himself with correction of manifest errors. But the manifest error is but a symptom. It is not the cause of the distress but a sign of the cause. We must learn to look for causes, remove causes, cease the treatment of symptoms.

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A FEW NOTES ON HYPERPHORIA, MYOPIA, PUPILLARY CONTRACTION AND THE CILIARY MUSCLE

BY R. M. PECKHAM*

Hyperphoria

The present writer has pointed out, in a volume on the use of the Kratometer, "Modern Treatment of Binocular Imbalances," that if the correction of an hyperphoria by vertical prisms, in either the position of "rest" or the position of "exercise," should increase an exophoria, the vertical prism could not be worn with comfort; while a vertical correction that would decrease an exophoria would prove very acceptable to the patient.

In our work at the Los Angeles School of Optometry, this past winter, our students have discovered another point that, it seems to me, is of greater value than the above note concerning the effect on the exophoria. For many times there is no appreciable immediate effect on the exophoria following the application of the vertical prism.

We find that that vertical prism, or combination of vertical

*[We welcome this opportunity to publish at this time, the above paragraphs by Dr. R. M. Peckham, one of the most outstanding research workers of our profession. This short manuscript and correction is the first technical communication he has written since leaving the Los Angeles School of Optometry.]—EDITOR, AMERICAN JOURNAL OF OPTOMETRY.

prisms, placed entirely over one eye, or divided between the two eyes, that increases the near abduction proves to be the correction gratefully accepted by the patient.

Sometimes the vertical prism that unlocks the tension will be placed base-up over one eye, sometimes base-down over the other eye; sometimes the amount is equally divided between the two eyes, sometimes an unequal division is necessary; too weak a prism sometimes fails to accomplish anything in the way of increased abduction; sometimes a weak prism will partially release the tension, pointing the way, then the addition of a bit more, sometimes over the same eye, sometimes in opposite direction over the other eye, will break the bonds and bring the desired abduction. So far, the general indications are that an overcorrection of the hyperphoria will again reduce the abduction, after the right amount has increased it. This latter point needs further study and verification.

Again, in cases of spurious hyperphoria, where the tonicity test with the maddox rod shows some hyperphoria but the vertical prism is rejected, we find that the base-in prism that increases the vertical ductions, making the comparison of base-down over the right eye more nearly equal to the duction with base-down over the left eye—that such a base-in prism will bring comfort to a sorely harassed pair of eyes. Sometimes such base-in prisms are equally divided between the two eyes, sometimes unequally divided, sometimes all the power is placed over one eye. We thought some connection might here be found showing the advisability of placing all the horizontal or vertical prisms either over the dominant or the non-dominant eye. But we fail to arrive at such a conclusion. For in some cases results are obtained by all the prism over the dominant eye, sometimes with all the power over the non-dominant eye.

The writer thinks that this is a subject worth considerable investigation and trial by practicing optometrists. If verification is made by a score or more of workers, with a number of cases aggregating several hundred, then we have indeed discovered something of extreme value in the handling of one of the most perplexing problems of binocular imbalances.

Myopia

More and more it appears that myopia is more a physiological problem than the purely optical problem it was for so long considered. Proper correction and treatment, it would seem, must be based on physiological principles, subordinating the optical neutralization of the apparent error. The trouble here is that so very little is known of the physiology of myopia. Three years ago the writer ventured the opinion that an understanding of myopia waited on fuller knowledge of endocrinology. That the solution of the mysteries of the endocrine glands would carry with it the answer to the riddle of myopia.

Now Sir Arthur Keith points to a possible, but, I think, only

partial, factor in the development of myopia. The overactivity, or imbalanced activity, of the endocrines, more particularly of the hypophosis, or pituitary body, controlling the growth of the fibroplastic cells, of which the sclerotic is composed, might be a possible cause of the excessive development of the eye globe. But this does not account for the phenomena of the first signs of "stretching" appearing at the limbus. Nor does it account for the hypertrophied meridional fibers of the ciliary muscles as found on the dissection of most eyes known to have been myopic. Nor does it account for the atrophy of the circular fibers of the ciliary muscles found in highly myopic eyes.

On the other hand, endocrine dysfunctions can and do cause many singular effects, both physically and psychically, and, if we but knew more of the anatomy and physiology of the fiber tracts, it might be that such effects as myopia could be directly traced to such dysfunctions. Still, would that be sufficient? Must not the work be carried further, to a point that will reveal the cause behind these dysfunctions?

It has been shown, I understand, that the circulation of blood in the myopic eye is not normal. Why? It has been shown, I believe this has been accepted, that the tissues, or some of the tissues, of the myopic eye are found deficient in certain calcerine salts. Why? This is traceable to dysfunctions in the endocrine system.

Maurinus, of Detroit, reports that in a study of a number of myopes he finds all with an involvement of the pituitary. This is work paralleling that of Keith. It would seem logical that more work should be done along this line.

I think we must arrive at the conclusion that there are several different types of myopia. These ought to be classified, if possible. For that treatment which is successful with one type is not so good for another type.

One of the most peculiar types is that in which minus lenses, either spherical or cylindrical, will not raise vision to normal but strong base-in prisms will. Scores of refractionists, both among the ophthalmologists and optometrists, are reporting these cases. Sometimes plano base-in prisms alone suffice, sometimes a small minus lens combination is needed, sometimes the full correction of the manifest myopia, when combined with the prism, will bring up vision. It is an odd characteristic of these cases that adding prism to a minus correction will not raise vision, but placing the prisms before the eyes and slowly building up the minus correction often brings vision to normal, or, at least, gives a marvelous improvement over what the minus correction alone will bring. Why? No one knows.

I trust I may be pardoned for here correcting a misstatement appearing in a review, in this Journal, January, 1929 issue, of the book, "Modern Treatment of Binocular Imbalances." The reviewer says, "He believes, but he is not sure, that the use of base-in, in

these cases, withdraws certain tension from the muscles thus increasing the circulation of blood to the retina, which may account for the improvement." But I "believe" nothing of the kind. Moreover, I doubt if such a sentence is sound physiology. At any rate, I should hate to have any physiologist think I ever conceived such an idea. This subject is nowhere presented in the book under review. It is unfortunate that the reviewer should have brought in extraneous matter.

I think the following is the paragraph the reviewer misconstrued. On page 122 of the booklet, "Lectures delivered at the Thirty-first Educational Congress of the American Optometric Association, held at Grand Rapids, Mich., July 23-25, 1928." following a discussion of the use of base-in prisms in myopia, appear these sentences: "We cannot understand why base-in prisms should give such a remarkable improvement in vision. One eminent ophthalmologist says, 'Tensions are removed, blood circulation is improved, of course vision is better.' But this does not seem sufficient reason. We want to know how and why the tensions are removed, how and why the blood circulation is improved."

The actual statement and its presentation by the reviewer are as far removed as the poles. I do not "believe," I doubt considerably, and only quoted the opinion of another, which he himself would wish to prove by laboratory methods before stating as a final conclusion.

The reader may have some interest in what suggested the thought our ophthalmologist friend uttered. It has been found, by laboratory methods, that the circulation of blood in the myopic eye is not normal, is, apparently, inadequate. Some have jumped to the hasty conclusion that it is this impairment of circulation that causes myopia. That we may reasonably doubt. Both are probably results of some occurrence farther back. It has been shown that the use of base-in prisms brings about curious relaxations in the sympathetic nervous system. Why, we do not know, nor can anyone guess how. But the effect is beneficial. And we have some evidence in a few cases of improved circulation of the blood, throughout the entire body. However, that is no proof, as yet, that this improvement can be directly traced to the inhibitions of the prisms. We must not be too hasty. We must abandon this habit of drawing conclusions from insufficient data, of assuming false premises and working up therefrom fallacious theories. Because a thing happens once, and under certain conditions, is no proof that it will happen again when a single variant introduces new conditions. Instead of "believing," personally I prefer disbelief, doubt, until such time as laboratory research shows more definite knowledge than at present obtains.

Meantime, we may use a working method that has been carefully tested by a number of thoughtful observers, knowing that its very use will help in bringing fuller knowledge. The one great thing needed by optometry and ophthalmology is unbiased, scientific re-

search. Practically all the theories of binocular imbalances that have appeared in the past have been founded on speculative philosophy, based on insufficient data or on the assumption that some previous theory has been proved true, while, to the contrary, there has been nothing in the way of actual work behind that original premise. Deductive logic can carry one to peculiar conclusions if the starting point is a false premise.

Pupillary Contraction

Anatomies give authoritatively certain pathways for the completion of the reflex arc of pupillary contraction in response to light. Where those definite statements came from originally we cannot say. They have been repeated over and over again in successive editions, copied by one author from another. A careful check back through all the anatomies ever published would be about the only way of determining the origin. But physiologists of the present day are only sure of one thing, that they do not know those pathways.

The course as originally premised runs from retinae to optic tracts, thence to external geniculate bodies, thence to the pregemina, where the afferent impulse was supposed to be transformed into efferent impulses, passing first to the nuclei of Edinger-Westphal, thence to the mesial nucleus of the IIIrd nerve, then via the IIIrd to the ciliary ganglia and so to the sphincter muscle.

Now it appears, from the work of several English and German anatomists, that the light reflex pathway leaves the optic tracts somewhere near their posterior ends and its subsequent course between the optic tracts and the ciliary ganglia is at present unknown.

The pathways given above as the original concept for the light reflex seem to be correctly charted for the visual reflex arcs, wherein pupillary contraction is associated in the combined convergence-accommodative act.

The original thought that pupillary contraction is an associate of accommodation does not now have much support. In the eighth edition of Dana's Nervous Diseases appears this sentence: "It is now thought that pupillary contraction is an associate of convergence rather than of accommodation." All reference to this is omitted in the ninth edition.

With the later idea that probably convergence-accommodation is a single function, an "attention reflex," utilizing the final common pathways from the mid-brain to the eye muscles but arriving at the mesenphalic center by its individual pathway, rather than by using the accommodative pathway and the convergence pathway in association, possibly one can conceive that the apparent hook-up between the convergence pathways, as noted in Dana's remark, and the pupillary visual contraction pathways, is, in reality, a union of pupillary contraction with the act of convergence-accommodation. Much more work needs to be done, it is evident, before any positive

statement can be made. Clinical evidence is that pupillary contraction is normally a part of the convergence-accommodation act, rather than of so-called accommodation, as is given in many text-books.

Indeed, it is somewhat doubtful if there is in the human adult, under normal conditions, an act of accommodation separate from and independent of convergence. As the testing methods used by refractionists in their routine office practice introduce artificial conditions, it is possible that such phenomena as "relative accommodation," "relative convergence," "fusion convergence," "lag of accommodation," etc., appear because of the patient's attempt to solve the dilemma introduced by these artificial conditions. Evidence is accumulating that seems to indicate that there are no such prenomena present under regular working conditions.

I do not believe, as I was quoted as having said in the review above mentioned, that pupillary contraction is an associate of convergence rather than of accommodation. I think there is enough evidence to form a working postulate, and a working postulate only, to be used until more definite information is obtained, that pupillary contraction is a normal part of, taking place with, the reflex of convergence-accommodation. As such a working postulate it helps in the diagnosis of many difficult cases of binocular dysfunctioning.

Once more, the need of research workers in the field of refraction is demonstrated. Such workers must have an ample background of laboratory training in physiology and psychology. Only thus can we combat the fallacies that guesswork has thrust upon us.

The Ciliary Muscles

Probably no term in our professional vocabulary is misused more than this: "The ciliary muscle." It is doubtful if there is a single muscle in the body about which less is known than this tiny collection of fibers whose activity is the most important subject of the refractionist's problem. Yet, to read and to listen to the statements concerning it, one would suppose that the writer of the text or he speaker on the platform is very well acquainted with its anatomy, innervation and physiology.

It would seem as though it might be more correct to say "The ciliary muscles." For this little body is really composed of three independent muscles, called, respectively, Circular Fibers, Radial Fibers, Meridional Fibers. From an anatomical study, we can assume that the Circular Fibers are used, or are contracted, in the act of accommodation, or focussing for an object nearer to us than the object we were originally looking at; that the Radial Fibers are used for relaxing the accommodation, that is, that they are contracted to flatten the crystalline lens when we change to a more distant object. Thus, it would seem that Circular and Radial are antagonistic, one extending when the other flexes. Then there would seem some logic in Tscherning's thought that the crystalline is held flat by tension. Had he used the term "Radial Fibers" rather than

"Ciliary Muscle," we could understand his theory better. But this idea is from anatomical study only. Physiological study has not been done, so far as the present writer has been able to learn.

The meridional fibers are a problem. From their anatomy we would assume that when shortened they pull the choroid forward, relax the ligaments supporting the crystalline, allow this to become convex. That looks as though they might partake in the normal act of accommodation. Their antagonist, for every muscle must have an antagonist, seems to be the elasticity of the choroid.

The innervation of the circular fibers seems to be over the third nerve. But what is the innervational pathway to the radial and meridional fibers? No one knows. By all normal logic, since the circular fibers are evidently innervated via a para-sympathetic route, the radial fibers should be innervated over the antagonistic route, the spinal sympathetic. But anatomical evidence is lacking.

In the dissection of eyes known to have been hyperopic, the circular fibers are found to be heavy, over developed, bundles, with the radial fibers correspondingly few and slender. In the dissection of eyes known to be myopic, the meridional fibers are heavy cables, with the circular fibers either atrophied or entirely absent.

Since the myope is demonstrably of the sympathetonic type, can it be that the over-development of the meridional fibers is a demonstration of the spinal sympathetic innervation of those fibers?

For many years physiologists have been trying to find a spinal sympathetic route, or connection in, the ciliary muscles. But instruments and technique are not yet sufficiently refined. Absence of proof of spinal sympathetic innervation to the radial or meridional fibers, or to both, cannot be accepted as positive proof that there is none, in the face of the overwhelming evidence from other sources that there is.

The ciliary muscles in man are of the involuntary, smooth, single cell type. But the third nerve, which then ought to be an involuntary nerve, has the microscopic appearance of a voluntary nerve. Then we have the anomaly, an involuntary muscle innervated through a voluntary nerve. What a physiological contradiction. Verily, there is much study needed.

But this apparently voluntary nerve preserves the methods of operation of an involuntary nerve. For, a voluntary nerve extends directly from its nucleus to the muscle it serves, while an involuntary nerve is made up of two parts: the "pre-ganglionic" fiber, from the nucleus in the brain stem or spinal cord to an outside ganglion, and the "post-ganglionic" fiber from this outside ganglion to the muscle. The portion of the third nerve serving the intrinsic muscles of the eyeball preserves this peculiarity of involuntary nerves, the pre-ganglionic fibers from the third nucleus terminating in the ciliary ganglia, the post-ganglionic fibers arising in the ciliary

ganglia and terminating in the muscles. This seems to be the only nerve of this sort in the entire body, a nerve resembling in structure the voluntary nerves, but functioning by the relay system of the involuntary nerves.

It would seem to behoove us to speak less lightly and less certainly of this muscle. It is hard to conceive of "spasm of accommodation" as a physiological possiblity. In latent hyperopia, we have, apparently, an hypertonicity or incomplete tetanus of the ciliary fibers instead of "spasm of the ciliary." In myopia, there seems to be a hypertonicity or incomplete tetanus of the meridional fibers rather than "spasm of accommodation." Then, to cap it all, in certain psychic reactions we get a short lived myopia. Whence comes that? Through the third nerve? It is hard to think it.

That there is an association between pupillary dilatation and innervation of the meridional fibers has been suggested, long ago. But it is not a provable concept. Now comes a bit of new light. Refer to your anatomies and you will see that the dilatator muscle of the iris is apparently without a point or origin. Berner found "The muscle is prolonged, in places by single cells, in places by groups of cells, with an accompanying connective tissue sheath resembling a tendon, which run obliquely into the ciliary body and are there prolonged to find some attachment to the pectinate ligament, thus providing the muscle with a fixed attachment." Thus our anatomies become antiques.

It is definitely known that the dilatator muscle of the iris is innervated through the spinal sympathetic system, via the ciliospinal center of Buge, in the cervical segment of the cord, through the superior cervical ganglion of the spinal sympathetic ganglionated cord. With this tracing of the dilatator muscle into the ciliary body, with its point of origin apposite to the point of origin of the meridional fibers, is their apparent associated innervation finally to be cleared up and demonstrated? Will this new knowledge, gained by improved technique and better instruments, serve as the point of attack through which the question of spinal sympathetic innervation to the ciliary fibers can be settled?

This may seem to some of our readers an academic question of doubtful practical value. But it is not. It is of supreme importance. For with this point definitely settled, the question of the value of atropine in refraction is forever solved. Nothing can be of more moment to the optometrist.

BOOK NOTICES

1929 YEAR BOOK OF THE NEW YORK STATE OPTOMETRIC ASSOCIATION. D. K. Uttal, Editor, B. J. Rosenkranz, Associate Editor, published by the New York State Optometric Association, 68 pages. Illustrated. Paper Covers, 1929. $1.00.

Thirteen technical papers are published in the 1929 Year Book of the New York State Optometric Association, in addition to a number of reports on Association activities. The technical papers are as follows: "Uncovering Latent Hyperopia," Dr. E. G. Bauman; "A Few Observations of Optometric Failures," Dr. J. O. Baxter; "Spherical and Chromatic Aberrations," Dr. Ben J. Campbell; "Application of Telescopic Spectacles," Drs. M. A. Silverman and Wm. Feinbloom; "The Case History," Dr. G. C. Farrell; "Two Special Sight Correcting Appliances," Dr. Victor M. E. Koch; "Suspenopsia," Dr. F. McFadden; "Myography," Dr. Wm. Arthur Mendelsohn; "Dynamic Skiametry, Accommodation and Convergence," Dr. S. Nott; "Romance of the Lens," Dr. W. B. Needles; "An Optometrist Looks at Philosophy," Dr. G. O. Ohlsson; "Hyperphoria from Correcting Lenses," Dr. Jos. I. Pascal, and "Subjective Application of Base-in Prism," Dr. Chas. I. Saks.

The book, as usual, is nicely gotten together and its editorial staff is again to be commended for the splendid work they have accomplished, however, we might suggest that in the future, all technical papers be placed together in one section so as to facilitate ready reference. This volume adds much to the prestige of the New York Association and being published as it is just prior to the Annual Convention, should go far towards assuring the officers a large attendance.

C. C. K.

OPHTHALMIC LENSES. H. H. Emsley, B. Sc., and Wm. Swaine, B. Sc., F. S. M. C. Published by the Hatton Press, Ltd., 78 Fleet Street, London, E. C. 4. 319 pages. 128 illustrations. 1929.

Ophthalmic Lenses by Emsley and Swaine is a valuable contribution to ophthalmic science and fills a gap in ophthalmic literature

which was long felt especially by the students, and this is just what the authors intended this book to accomplish.

The book is made up of seventeen chapters. The first twelve deal mostly with practical and applied optics; subjects are discussed which are daily applied either by the refractionist, dispenser, or shop man.

Chapters 1 to 8, inclusive, are devoted to the discussion of spectacle lenses, shape-sized and form. Prisms and bifocals are given special consideration. Chapter 9 is devoted to protective glasses. Here, the authors show a broad knowledge of the subject of raidant energy and its effect upon the refractive system of the eye, suggesting certain types of tinted lenses to be prescribed for specific conditions, and chapters 10 and 11, to optical dispensing as well as to the description of surfacing and lens grinding machines and their use. Chapter 12 deals with lens forms and thickness, as well as effective and vertex power. The last five chapters describe certain types of lens forms which are of interest to manufacturers and instrument designers.

Each chapter is followed by a group of exercises and problems, the answer to these is given in the appendix. In this part is also given a considerable number of tables of measures, trigonometric functions, refractive indicies and many others which are of value as a ready reference guide to the student as well as the practitioner. This book "Ophthalmic Lenses" contains valuable information and will find a welcome place in the libraries of the studious optometrists of the United States as well as in England.

J. I. K.

ANNOTATIONS AND SOCIETY PROCEEDINGS

The American Journal of Optometry is the official Organ of the American Academy of Optometry, as well as the Organ of the Arkansas, Delaware, Iowa, Maine, Maryland, Michigan, Minnesota, Nebraska, North Dakota, Oklahoma, Pennsylvania, South Dakota, Vermont and West Virginia Optometric Associations.

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SOUTH DAKOTA OPTOMETRIC CONVENTION

The South Dakota Optometric Convention was held at the Marvin Hughitt Hotel at Huron, S. D. According to Dr. S. L. Kirkpatrick, an interesting and educational program was arranged for by the convention committee. Dr. Chas. Sheard of the Mayo Clinic delivered two lectures, one on "The Laws of Supply and Demand in Problems of Ocular Refraction" and the second lecture was on "The Zones of Ocular Comfort." Dr. Jack Copeland lectured on "Dynamic Retinoscopy and its Interpretations" and also on "The Value of Field Studies to Optometrists."

The following officers were re-elected: Dr. H. N. Smith, President; Dr. W. H. Fritz, Secretary; Dr. A. F. Rounds, Treasurer. President Smith was unanimously elected as delegate to represent the State Association at the A. O. A. Convention. Dr. Fritz was elected as the alternate. The Association voted to continue the American Journal of Optometry as its official publication and to have copy of the Journal sent to the entire paid up membership—the cost to be paid for from the Association Funds.

The Convention closed with a very elaborate banquet. Dr. E. G. Colberg acted as the Toastmaster. Bids for the next annual convention were received from Aberdeen, Pierre, Rapid City and Mitchell. It was voted unanimously to hold the next convention at Mitchell during the last half of the month of April, 1930.

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WEST VIRGINIA ANNUAL CONVENTION

The Annual Convention of the West Virginia Association of Optometrists was held at the Chanceler Hotel, Parkersburg, on Monday, April 22nd. According to Dr. John T. Collins, the convention was well attended and the Graduate Work conducted by Dr. A. F. Skeffington was decidedly outstanding. A free clinic was also held during the convention period.

Dr. L. G. Stortz was re-elected President; Dr. W. W. Brown was elected First Vice President; Dr. J. R. Koch, Second Vice President; Dr. John T. Collins, Secretary, and Dr. C. M. King, Treasurer. The Executive Committee will consist of the above named gentlemen in addition to the following: Dr. D. L. Gallagher, Dr. C. C. Wilson, Dr. J. Frank Brown and Dr. G. G. Mead.

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A. O. A. GOLF TOURNAMENT

Optometrists attending the Annual Convention of the A. O. A., the week of June 17th, in Philadelphia, should arrange to participate in the convention golf tournament sponsored by that organization. The tournament will be held at the Roxboro Country Club and a number of interesting prizes have been set up by the committee in charge. Optometrists wishing to enter this tournament should apply to Dr. J. D. Shoptaugh, 213 S. Eighth St., Philadelphia, Pa.

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OKLAHOMA BOARD OF EXAMINERS

Dr. E. B. Alexander, Secretary of the Board of Examiners in Optometry for the State of Oklahoma, announces that the next examination conducted by this Board will be held at the Huckins Hotel, Oklahoma City, Oklahoma, on July 24th, 25th and 26th, 1929. Application should be made to Dr. E. B. Alexander, Duncan, Okla.

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MAINE ASSOCIATION MEETS

The Maine Association of Optometrists met at Farmington, on May 22nd. Dr. Sam Baker presented a technical paper on "The Cross Cylinder," and a report on "Clinical Work." Dr. Philip Dean read a paper on the "Clinical Value of Relative Accommodation and Convergence."

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EDUCATIONAL PROGRAM A. O. A. CONGRESS

Dr. Albert Fitch, President of the Pennsylvania State School of Optometry and Chairman of the Educational Committee for the coming Convention of the A. O. A. to be held at Philadelphia, June 17th to 22nd, announces that the educational program to be conducted during that week will be as follows: first, a detailed series of demonstrations covering every phase of optometric diagnostic and refractive work and, second, a series of

lectures dealing with optometric jurisprudence, optometric economics, office practice and illumination. This appears to be one of the fullest and most comprehensive educational programs staged by the A. O. A. in the last decade and the committee is arranging to handle a large group of practitioners. The majority of the educational work will be held at the Pennsylvania State College of Optometry.

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MINNESOTA STATE BOARD — Dr. Ernest Kiekenapp, Secretary of the Minnesota State Board of Optometry, announces that the next examination of this Board will be held at the old State Capital Building, St. Paul, Minnesota, on July 10th and 11th.

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VANDERBURGH ACADEMY OF OPTOMETRY — The Vanderburgh Academy of Optometry held its annual meeting with dinner at Hadi Temple, March 8th. A resume of year's work showed much accomplished along educational and inspirational lines. During the year the Academy has listened to lectures from several of the outstanding men of Optometry such as Packham, Skeffington and Shepard and this has greatly increased the interest in our work.

The election of officers resulted in re-electing Dr. C. V. Waters, president; Dr. J. Royston Victor, vice-president, and Dr. M. O. Cockrum, secretary and treasurer.

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